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EDITORIAL COMMENT

Contrast-Enhanced CMR Imaging of Ventricular Tachycardia Isthmus Sites to Guide Ablation

An Approach in Evolution*

Srinivas R. Dukkipati, MD, Javier Sanz, MD

The mechanism of ventricular tachycardia (VT) in patients with structural heart disease is primarily scar-related reentry (1). Although any disease process that results in the formation of myocardial scar can predispose to reentrant VT, the best characterized substrate is post-myocardial infarction VT. After myocardial infarction, ventricular tissue can be classified into 3 types: normal myocardium, dense scar, and the intervening border zone (BZ). In the BZ, myocardial fibrils are interspersed between electrically inert fibrotic tissue causing electrical conduction to take a circuitous path and, together with abnormal myocyte cell-to-cell coupling, causes slow conduction, a necessary component for reentrant VT (2). Electrical conduction through these myocardial channels can exit the scar and depolarize normal myocardium resulting in 12-lead electrocardiogram morphology of the VT that is dependent on the location of this exit site. Myocardial channels are identified during VT ablation on the basis of their abnormal conduction properties, which yield fractionated and late potentials and are incorporated in the ablation strategy based on observations that they are strongly associated with VT isthmus sites (3). Additionally, pacing from within the BZ may identify a potential VT isthmus site when the paced 12-lead QRS morphology matches the VT. Although late potentials and good pacemapping sites may denote potential VT isthmus sites, their participation in any VT can only be proven by entrainment maneuvers performed during tachycardia and by termination of VT during ablation at the site (4). However, only a fraction of patients will have exclusively hemodynamically tolerated VT to allow detailed entrainment mapping. Therefore, a substrate-based ablation strategy during sinus or paced rhythm is commonly used. This approach uses a 3-dimensional electroanatomic mapping system that allows: 1) spatial localization of a mapping catheter; 2) construction of a 3-dimensional anatomic representation of a cardiac chamber based on bipolar voltage information recorded from the catheter tip to allow delineation of normal and infarcted myocardium; and 3) cataloging of potential VT isthmus sites identified by brief entrainment, the presence of late potential, or good pacemapping sites that are then targeted for ablation (5). This process is not only tedious and time-consuming, but electroanatomic mapping provides a rather limited picture of the true extent of myocardial scar that is restricted to the surface being mapped (i.e., the endocardial or epicardial surface). Information regarding the extent of intramyocardial scar or scar on the opposing surface is not obtained. Whether cardiac magnetic resonance (CMR) can simplify this process by providing detailed information regarding myocardial scar architecture is under active investigation.

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In this issue of *iJACC*, Piers et al. (6) describe the CMR characteristics of critical isthmus sites in 44 patients with ischemic and nonischemic cardiomyopathy undergoing VT ablation. Critical isthmus sites for 78 of 110 inducible VTs were identified based on pacemapping in 67, concealed entrainment in 10, and arrhythmia termination in 31 VTs. Critical sites were then projected on 3-dimensional scar reconstructions derived from pre-procedural CMR, and local late gadolinium enhancement (LGE)

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From the Icahn School of Medicine at Mount Sinai, New York, New York. Dr. Dukkipati is a consultant for Biosense Webster, Inc. Dr. Sanz has reported that he has no relationships relevant to the contents of this paper to disclose.

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characteristics were analyzed. These included transmurality, density (scar core and BZ defined as regions with signal intensity >50% and between 35% and 50% of maximal myocardial signal intensity, respectively), distance to the core-BZ transition, and distance to areas with scar transmurality >75%. Critical ablation sites were located within 5 mm of >75% transmural scar in 74% to 100% of cases, depending on the mapping criteria used, and within 5 mm of the core-BZ zone in 67% to 100%. Areas fulfilling both CMR criteria contained all isthmus sites defined by concealed entrainment, 56% of those defined by pacemapping, and 77% of VT termination sites.

The authors are to be congratulated for performing a meticulous characterization of scar architecture, as determined by LGE, of critical sites for VT ablation in a sizable number of patients with both ischemic and nonischemic cardiomyopathies. They demonstrate that critical isthmus sites are often located in the vicinity of areas with high degrees of scar transmurality, yet also limited scar density. Although these findings may seem contradictory, they certainly are not. Although the misleading term BZ may suggest a region located at the scar periphery simultaneously containing dense scar adjacent to normal myocardium, the CMR definition most often used based on signal intensity rather than transmurality is more reflective of interspersed fibrotic and viable tissue across part or all of the myocardial thickness. Given the spatial heterogeneity of ischemic and nonischemic scar, its characterization as a region with varying degrees of fibrosis transmurality and density throughout seems much more realistic than an oversimplified view of a compact core surrounded by less dense fibrosis. Then, the presence of BZ areas in any location within the scar, including the core, is not unexpected. Indeed, previous studies integrating LGE and electroanatomic mapping have shown that conducting channels and/or critical VT circuit regions often correspond to areas with intermediate signal intensity located within the near-transmural core of the scar (7–11). This is in line with the findings of the present study of a predominant clustering of critical isthmus sites near areas with >75% transmurality and/or the core-BZ transition. This information may aid ablation procedures by guiding mapping preferentially to such regions if scar architecture is known in advance. One important question yet remains unanswered, specifically, the diagnostic performance of the criteria proposed by the authors for differentiating critical from noncritical intrascar sites. Even though regions within 5 mm of >75% transmural scar and/or the core-BZ interface are relatively confined (i.e., only a median of 13% of the left ventricular area in the present study fulfilled both criteria), they still represent a substantial percentage of the total scar area (median, 24% of the left ventricle). Thus, it is likely that many intrascar noncritical areas also demonstrate one or both features. A comparison of the CMR characteristics of critical isthmus sites with randomly selected, noncritical regions would strengthen the study and help to answer this question, and we certainly encourage the authors to perform such an investigation.

This study further illustrates the potential role of CMR to guide electrophysiological interventions. Before it becomes a mainstream tool, consistency in the methodology for scar core and BZ quantification will be needed. Previous definitions include the one used by the authors using thresholds of 50% and 30% of maximal signal intensity (12) or 60% and 40% (10), or 3 and 2 SDs above normal myocardial signal (13). It is also possible that performance may improve with individualized threshold settings (10). Furthermore, signal intensity is not only determined by tissue characteristics but also by imaging variables such as spatial resolution, contrast dose, and timing of acquisition. With further standardization of imaging protocols, CMR may perhaps facilitate identification of VT critical isthmuses in clinical practice, which could result in reduced procedural duration and/or an increased success rate.

REPRINT REQUESTS AND CORRESPONDENCE: Dr. Srinivas R. Dukkipati, Icahn School of Medicine at Mount Sinai, One Gustave L. Levy Place, Box 1030, New York, New York 10029. E-mail: srinivas.dukkipati@mountsinai.org.

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